Diagnosis of the Metabolic Syndrome Is Associated With Disproportionately High Levels of High-Sensitivity C-Reactive Protein in Non–Hispanic Black Adolescents

An analysis of NHANES 1999–2008

MARK D. DEBOER, MD, MSC, MCR[1]
MATTHEW J. GURKA, PHD[2]
ANNE E. SUMNER, MD[3]

OBJECTIVE—Whereas it is known that the metabolic syndrome (MetS) has a paradoxically lower prevalence in non–Hispanic black adolescents than in non–Hispanic whites or Hispanics, the relative severity of MetS by race/ethnicity is unknown. Inflammation, indicated by high-sensitivity C-reactive protein (hsCRP), is a key factor linking MetS to cardiovascular disease and type 2 diabetes. Our goal was to determine whether elevations of hsCRP vary by race/ethnicity among adolescents with MetS.

RESEARCH DESIGN AND METHODS—We used the National Health and Nutrition Examination Survey (1999–2008) and evaluated adolescents (age 12–19 years) using a pediatric/adolescent adaptation of the ATP III definition of MetS. We used linear regression to evaluate the interaction between MetS status and ethnicity with respect to hsCRP concentration.

RESULTS—For male and female adolescents, MetS was associated with elevated hsCRP levels compared with adolescents without MetS. However, the elevation in hsCRP between adolescents with and without MetS was greater in non–Hispanic blacks compared with that in non–Hispanic whites ($P = 0.04$) but not that in Hispanics ($P = 0.18$). hsCRP concentrations correlated with individual MetS components similarly among all ethnicities. In an evaluation of adolescents diagnosed with MetS, non–Hispanic blacks had higher BMI and more hypertension than other ethnicities but there were no other racial/ethnic differences in the features of MetS.

CONCLUSIONS—Non–Hispanic black adolescents have a greater differential in hsCRP between those with and those without MetS than the differential in non–Hispanic whites but not that in Hispanics. Therefore, even though MetS has a low prevalence in non–Hispanic blacks, MetS is a particularly good indicator of inflammation in non–Hispanic black adolescents.

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The metabolic syndrome (MetS) is a clustering of cardiovascular risk factors that are related to insulin resistance, specifically, elevated waist circumference, hypertriglyceridemia, low HDL-cholesterol, hypertension, and fasting hyperglycemia (1). MetS is related to inflammation (2) and functions as an independent predictor of long-term risk for cardiovascular disease and type 2 diabetes among both adults (3) and children (4). Whereas the relationships between MetS, inflammation, and long-term risks are well described in the general population, these relationships have not been delineated in non–Hispanic black individuals. Non–Hispanic blacks are less likely than non–Hispanic whites and Hispanics to be classified as having MetS, largely based on a lower prevalence of dyslipidemia (5–7). Nevertheless, non–Hispanic blacks are more likely than non–Hispanic whites to exhibit insulin resistance (8,9) and to develop cardiovascular disease and type 2 diabetes (10,11), which brings into question the accuracy of current criteria for classifying MetS among non–Hispanic blacks (6). Similarly, the relationship of MetS with increased inflammation among non–Hispanic blacks is unclear.

High-sensitivity C-reactive protein (hsCRP) is a marker of inflammation that in adults is an independent risk factor for cardiovascular disease and type 2 diabetes (12,13). Among children and adolescents, elevated hsCRP levels are predictive of adult hsCRP levels 20 years later (14) and are independently associated with arterial changes that precede cardiovascular disease, including carotid artery intima-media thickness (15).

Our goal was to determine whether elevations in hsCRP values among adolescents with MetS vary by race/ethnicity. We used the National Health and Nutrition Examination Survey (NHANES) 1999–2008 and a pediatric/adolescent adaptation of the Adult Treatment Panel III (ATP III) definition of MetS (1) to explore the relationship between MetS and hsCRP on a race/ethnicity-specific basis.

RESEARCH DESIGN AND METHODS

—Data were obtained from NHANES (1999–2008), a complex, multistage probability sample of the U.S. population. These annual surveys are conducted by the National Center for

From the [1]Department of Pediatrics, University of Virginia, Charlottesville, Virginia; the [2]Department of Community Medicine, West Virginia University, Morgantown, West Virginia; and the [3]National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, Maryland.
Corresponding author: Mark D. DeBoer, deboer@virginia.edu.
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Health Statistics of the Centers for Disease Control, with data released every 2 years (http://www.cdc.gov/nchs/nhanes.htm). The National Center for Health Statistics ethics review board approved the survey, and participants were provided with informed consent prior to participation. As previously described, waist circumference, blood pressure, and laboratory measures of triglycerides, HDL-cholesterol, and glucose were obtained using standardized protocols and calibrated equipment (1). All blood samples used for analyses were obtained from participants asked to fast ≥8 h prior to blood draw.

Diagnosis of MetS

MetS was defined by a pediatric/adolescent adaptation of the ATP III criteria (1). Participants had to meet three or more of the following five criteria: concentration of triglycerides ≥110 mg/dL, HDL-cholesterol ≤40 mg/dL, waist circumference ≥90th percentile for age/sex (or, if lower, the ATP III limit of 102 cm for male participants and 88 cm for female participants) (16), glucose concentration ≥100 mg/dL, and systolic or diastolic blood pressure ≥90th percentile (age, height, and sex specific) (17).

Data from non–Hispanic white, non–Hispanic black, or Hispanic (Mexican American/other Hispanic) adolescents age 12–19 years were analyzed. Children <12 years old were excluded because fasting values for triglycerides and glucose were only obtained in participants ≥12 years old. Subjects with known diabetes and pregnancy and individuals taking antihyperlipidemic or antidiabetic medications were excluded from the study. Participants taking antihypertensive medication were classified as having hypertension. Children with hsCRP ≥10 mg/L were excluded because of an association with acute infection or chronic inflammatory disease (18).

Statistical analysis

Statistical significance was defined as a *P* value ≤0.05. To maximize total sample size, we combined datasets from the four 2-year cycles (1999–2008) for statistical analyses. Prevalence rates of MetS were calculated by sex, race/ethnicity, and NHANES cycle and compared with χ^2 tests. Mean hsCRP was compared among groups using unpaired Student *t* tests or ANOVA. Linear regression was used to assess the effect of sex, race/ethnicity, and MetS status on levels of ln (hsCRP). The natural log transformation

Table 1—*NHANES 1999–2008 characteristics for subjects 12–19 years old with data on all outcomes of interest* (n = 3,559)

	n	With Mets (%)	BMI (kg/m^2)	Waist (cm)	Triglycerides (mg/dL)	HDL-cholesterol (mg/dL)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Fasting glucose (mg/dL)	hsCRP (mg/L)
Overall	3,559	8.4	23.3 (23.0–23.6)	80.9 (80.1–81.6)	90.1 (87.2–93.1)	51.0 (50.4–51.6)	109.7 (109.0–110.4)	61.9 (61.4–62.5)	93.3 (92.8–93.8)	1.1 (1.0–1.2)
Sex										
Male	1,887	11.0	23.2 (22.8–23.6)	81.5 (80.4–82.6)	92.1 (88.2–96.0)	48.9 (48.1–49.7)	112.5 (111.6–113.4)	60.6 (59.9–61.4)	95.1 (94.6–95.6)	0.9 (0.9–1.0)
Female	1,672	5.5	23.3 (23.0–23.7)	80.2 (79.2–81.1)	88.1 (84.1–92.1)	53.2 (52.4–54.1)	106.7 (105.9–107.5)	63.3 (62.6–64.0)	91.4 (90.6–92.1)	1.2 (1.1–1.3)
P†		<0.01	0.61	0.05	0.14	<0.01	<0.01	<0.01	<0.01	<0.01
Ethnicity										
Non–Hispanic white	1,018	8.6	22.9 (22.6–23.3)	80.7 (79.6–81.8)	93.6 (89.7–97.4)	50.2 (49.4–51.1)	109.4 (108.5–110.3)	62.4 (61.7–63.2)	93.4 (92.8–94.0)	1.0 (0.9–1.1)
Hispanic	1,428	10.8	23.8 (23.3–24.3)	82.3 (81.0–83.6)	95.2 (88.9–101.5)	50.3 (49.5–51.2)	109.2 (108.2–110.2)	60.4 (59.6–61.1)	94.5 (93.6–93.8)	1.4 (1.2–1.5)
Non–Hispanic black	1,113	4.5	24.3 (23.9–24.7)	79.9 (78.9–80.8)	68.4 (66.0–70.9)	55.4 (54.4–56.4)	111.8 (111.1–112.5)	61.6 (60.6–62.5)	91.3 (90.7–91.8)	1.1 (1.0–1.2)
P‡		<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Year										
1999–2000	817	8.1	23.0 (22.6–23.5)	79.9 (78.7–81.2)	89.4 (83.7–95.0)	48.0 (46.7–49.2)	110.3 (109.3–111.3)	64.8 (63.9–65.6)	92.0 (91.4–92.7)	1.1 (0.9–1.3)
2001–2002	824	9.2	22.8 (22.4–23.2)	79.5 (78.3–80.8)	91.7 (85.7–97.6)	48.5 (47.8–49.3)	108.6 (107.0–110.1)	63.0 (62.2–63.8)	94.2 (93.5–95.0)	1.1 (0.9–1.2)
2003–2004	782	8.0	23.4 (22.8–24.0)	81.6 (80.1–83.1)	92.2 (84.0–100.4)	52.3 (51.3–53.4)	109.5 (108.2–110.7)	60.4 (59.1–61.7)	92.4 (91.1–93.7)	1.1 (1.0–1.3)
2005–2006	754	9.6	23.5 (22.7–24.3)	81.9 (79.5–84.3)	91.5 (84.1–98.9)	52.9 (51.6–54.3)	110.7 (108.6–112.7)	60.7 (59.1–62.3)	93.1 (92.0–94.3)	1.1 (0.9–1.3)
2007–2008	382	6.9	23.6 (22.9–24.3)	81.3 (79.5–83.1)	86.0 (80.3–91.8)	53.0 (51.2–54.7)	109.7 (108.2–111.2)	61.2 (59.6–62.7)	94.3 (92.8–95.8)	1.0 (0.9–1.2)
P‡		0.87	0.21	0.14	0.62	<0.01	0.36	<0.01	<0.01	0.81

Data are means (95% CI) unless otherwise indicated. †χ^2 test comparing % with MetS, *t* test comparing mean values of MetS components; ‡χ^2 test comparing % with MetS, ANOVA comparing mean values of MetS components (overall difference among the groups).

of hsCRP was used to achieve normality. All interactions of the three covariates (sex, race/ethnicity, and MetS status) were initially included in the model but removed in a stepwise fashion if the associated interaction *P* value was >0.15. Because of the known effects of poverty (19), education (19), and smoking (20) on hsCRP, each of these covariates was included in the model. Education was classified as the highest level obtained for any household member and categorized as follows: less than high school, high school, and more than high school. Income-to-need ratio was used to measure poverty. Because of the poor reliability of self-reporting of smoking among adolescents (21), serum level of cotinine was used to identify smokers, with a cutoff of 15 ng/mL as recommended (22). Geometric means of hsCRP from the final model were estimated and compared among race/ethnicity, as applicable.

Following the comparison of geometric means of hsCRP, racial/ethnic differences in the relationship between hsCRP and MetS were further evaluated. This was performed by calculating Pearson *r* correlation coefficients to determine the degree of linear association between MetS components and ln(hsCRP) by race/ethnicity. Finally, means and prevalence of MetS components were compared by race/ethnicity among adolescents with MetS. With the exception of the correlation estimates, all analyses incorporated the sampling weights included from NHANES. Statistical analysis was performed using SAS (version 9.2; SAS, Cary, NC) and SUDAAN (version 10; Research Triangle Institute, Research Triangle Park, NC), which account for the survey design when estimating SEs to obtain population-based estimates.

RESULTS

MetS prevalence

The sample of participants consisted of 3,559 non–Hispanic blacks, non–Hispanic whites, and Hispanics age 12–19 years with data for all variables tested. Among adolescents, the prevalence of MetS was greater among male than female participants (11.0 vs. 5.5%; $P < 0.01$) and among non–Hispanic whites and Hispanics compared with non–Hispanic blacks (8.6, 10.8, and 4.5%, respectively; $P < 0.05$) (Table 1), as has previously been reported for adolescents and adults (5,6). The prevalence of MetS did not differ over the test period (1999–2008).

Individual components of MetS are presented in Table 1. Compared with non–Hispanic whites and Hispanics, non–Hispanic blacks had lower triglycerides and fasting glucose and higher HDL-cholesterol and systolic blood pressure. Hispanics had the highest waist circumference but otherwise did not exhibit differences in individual MetS components compared with non–Hispanic whites and non–Hispanic blacks.

hsCRP levels

Levels of hsCRP were higher in adolescent female than in male participants. hsCRP levels were also higher in Hispanics than in non–Hispanic whites and non–Hispanic blacks (Table 1).

Covariates in the linear model of ln (hsCRP) are shown in Table 2. Household education level and smoking were significantly associated with levels of hsCRP, as shown previously (19,20). Two pairwise interactions were significant and thus remained in the model: sex × ethnicity ($P = 0.0494$) and MetS × ethnicity ($P = 0.1249$). Overall, female participants had higher hsCRP values than male participants (13), with the greatest difference between sexes observed among non–Hispanic blacks. MetS had a significant effect on hsCRP for all examined ethnicities, with hsCRP levels increasing the most for non–Hispanic blacks with MetS.

Geometric means of hsCRP levels by sex and ethnicity (generated from the linear model of ln(hsCRP)) are shown in Fig. 1*A–B*. In both male and female participants, each ethnicity exhibited higher hsCRP values among adolescents with MetS compared with those in adolescents without MetS ($P < 0.01$ for all three ethnicities). Among adolescent male participants without MetS, Hispanics had higher levels of hsCRP than non–Hispanic blacks ($P = 0.017$) but not non–Hispanic whites ($P = 0.10$). For adolescent female participants without MetS, Hispanics had higher levels of hsCRP than non–Hispanic whites ($P < 0.01$), whereas hsCRP levels in non–Hispanic blacks were not significantly different than those in non–Hispanic whites ($P = 0.05$). Among male participants with MetS, there were nonsignificant trends toward higher hsCRP levels in non–Hispanic blacks ($P = 0.07$) and Hispanics ($P = 0.08$) than in non–Hispanic whites. Among adolescent female participants with MetS, both non–Hispanic blacks and Hispanics had higher levels of hsCRP than those found in non–Hispanic whites ($P < 0.01$ and $P = 0.02$, respectively). The difference in hsCRP (as expressed by the ratio of geometric means of hsCRP) between adolescents with MetS and those without was greater in non–Hispanic blacks than in non–Hispanic whites ($P = 0.04$) but not

Table 2—*Linear model results of ln(hsCRP)

Model covariate	Estimate	95% CI	*P*
Intercept	−0.97	−1.13 to −0.81	<0.01
Education†			
Less than high school	0.20	0.08–0.32	<0.01
High school	−0.01	−0.19 to 0.18	0.94
Income-to-needs ratio	−0.02	−0.06 to 0.01	0.21
Current smoker	0.22	0.06–0.38	<0.01
Race/ethnicity‡			
Hispanic	0.16	−0.03 to 0.35	0.09
Non–Hispanic black	−0.09	−0.24 to 0.07	0.27
Female§			
Non–Hispanic white	0.15	0.03–0.28	0.02
Hispanic	0.28	0.06–0.51	0.01
Non–Hispanic black	0.44	0.23–0.64	<0.01
MetS¶			
Non–Hispanic white	1.04	0.82–1.26	<0.01
Hispanic	1.19	0.90–1.47	<0.01
Non–Hispanic black	1.56	1.11–2.01	<0.01

*Final model included sex × ethnicity interaction ($P = 0.0494$) and MetS × ethnicity interaction ($P = 0.1249$); model $R^2 = 0.086$. †Highest among household (person who owns/rents house or his/her spouse); values indicate difference from "more than high school" category. ‡Values indicate difference from non–Hispanic whites. §Values indicate difference from male subjects for corresponding ethnic group. ¶Values indicate difference from non–MetS individuals for corresponding ethnic group.

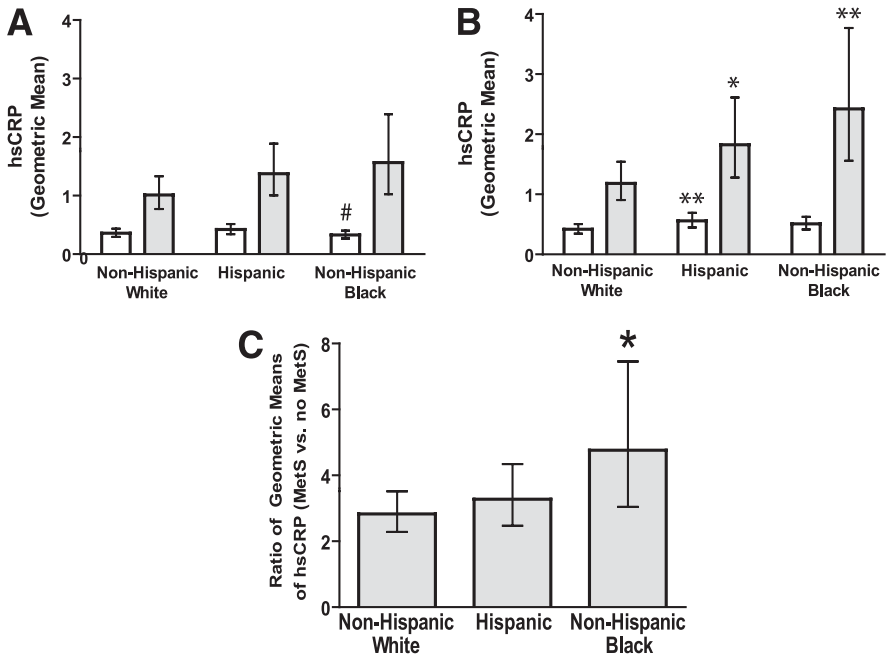

Figure 1—*Comparison of hsCRP concentrations by ethnicity.* A *and* B*: Adjusted geometric means of hsCRP by sex, ethnicity, and MetS status. Estimated geometric means (95% CIs) for male* (A) *and female* (B) *participants among adolescents with (white) and without (gray) MetS, for nonsmokers with a high school degree and an income-to-needs ratio of 2.* C*: Ratio of adjusted geometic means (95% CIs) of hsCRP values ($MetS^+/MetS^-$) by ethnicity. Male and female subjects were combined because of a lack of an interaction between MetS, ethnicity, and sex (i.e., the ratio of $MetS^+$ to $MetS^-$ by ethnicity was constant for male and female subjects). For* A–C*, comparisons between ethnic groups by corresponding MetS status are as follows:* *$P < 0.05$ *and* **$P < 0.01$ *vs. non–Hispanic whites;* #$P < 0.05$ *vs. Hispanics.*

higher than the difference in Hispanics ($P = 0.18$) (Fig. 1*C*).

Correlations of hsCRP with individual MetS components

One possible explanation for higher levels of hsCRP among non–Hispanic black adolescents with MetS is that hsCRP is more tightly linked to the components of MetS than is seen among other ethnicities. Therefore, to evaluate qualitative relationships between hsCRP and MetS among each race/ethnicity and sex, we assessed correlations of ln(hsCRP) with BMI and individual MetS components (Table 3). With the exception of diastolic blood pressure and fasting glucose, hsCRP was significantly correlated with each of the components of MetS in each of the sex/ethnicity groups, with strengths of association that were similar between non–Hispanic blacks and the other ethnicities.

Comparison of individual MetS components among adolescents with MetS

Another potential explanation for a greater differential in hsCRP levels among non–Hispanic black adolescents is that non–Hispanic blacks may not be classified as having MetS until they have a more developed condition. To investigate this, we determined mean values for MetS components among individuals with MetS, along with the proportion above/below the MetS-defining cutoffs of these components (available in the Supplementary Materials). Non–Hispanic black adolescents with MetS had a higher BMI but not waist circumference than either other group. Non–Hispanic blacks with MetS had a higher mean systolic blood pressure than did Hispanics, and there was a higher prevalence of hypertension among non–Hispanic blacks with MetS than among either other group. There were no other significant differences in MetS components between groups.

CONCLUSIONS—We report for the first time that non–Hispanic-black adolescents with MetS have higher levels of hsCRP than non–Hispanic whites but not Hispanics and have a greater differential in hsCRP between individuals with MetS and those without MetS. These data may indicate that non–Hispanic black adolescents with MetS have a more advanced inflammatory condition than non–Hispanic whites. Therefore, even though MetS has a paradoxically low prevalence in non–Hispanic blacks, MetS may be a particularly good indicator of inflammation in non–Hispanic black adolescents.

Although the value of MetS has been challenged as an entity that is more meaningful than the sum of its parts (23), the associations between the individual components of MetS are powerful, as is the strong relationship between MetS and systemic inflammation (2). Indeed, inflammation is frequently cited as a key etiologic factor in the development of the individual components that comprise MetS (2).

In our analysis of NHANES data, we considered several explanations that could contribute to the higher elevation in hsCRP in non–Hispanic black adolescents with MetS than in non–Hispanic whites. First, there may be environmental characteristics—such as socioeconomic or educational differences (19)—that could impact hsCRP levels differently by race/ethnicity. To address these possibilities, we included adjustment for household income, education, and smoking status in our linear model (Table 2). Although education and smoking were clearly linked to hsCRP levels, adjusting for differences in these covariates did not eliminate the significant effect among non–Hispanic blacks. Nevertheless, the possibility of residual confounding cannot be discounted because other factors related to hsCRP and ethnicity could in theory account for our findings.

A second possibility is that an elevated degree of inflammation may represent a unique feature of the processes underlying MetS among non–Hispanic blacks—e.g., that non–Hispanic blacks exhibit an exaggerated production of inflammatory markers from adipose tissue or elsewhere. This theory is plausible because there are many characteristics of obesity and MetS that manifest differently in non–Hispanic blacks than in non–Hispanic whites and Hispanics. The most striking of these ethnic differences relates to lower rates of dyslipidemia among non–Hispanic blacks (5,6). Additionally, compared with non–Hispanic whites, non–Hispanic blacks have greater insulin resistance at similar levels of adiposity (8,9) and less visceral adiposity even after adjustment for total fat and waist circumference (8,24). Non–Hispanic blacks also have more subcutaneous fat than non–Hispanic whites and more lean body mass per unit of body

weight (24). Also, compared with both non–Hispanic white and Hispanic adolescents, non–Hispanic blacks are less likely to exhibit elevated fasting blood glucose values (6) despite their higher incidence of type 2 diabetes (10). Therefore, the exaggerated levels of hsCRP among adolescents with MetS may represent an additional unique feature of MetS in non–Hispanic blacks.

Consequently, we analyzed linear relationships between hsCRP and each of the individual components of MetS. In all three racial/ethnic groups, hsCRP correlated similarly with each of the components of MetS (Table 3), suggesting that there are not qualitative differences in the relationship between hsCRP and MetS among non–Hispanic blacks. However, the potential remains for more subtle variation in the relationship between hsCRP and the processes underlying MetS.

Finally, we investigated whether the higher elevation in hsCRP among non–Hispanic blacks with MetS is due to the underclassification of MetS among non–Hispanic black adolescents. It has been well documented that non–Hispanic blacks are classified as having MetS at far lower rates than are seen among non–Hispanic whites and Hispanics (5,6). These low rates of MetS classification are at odds with the greater degree of insulin resistance (8,9) and high rates of type 2 diabetes and cardiovascular disease in non–Hispanic blacks (10,11). One of the most likely etiologies of the underclassification of MetS is the relative absence of the dyslipidemia of insulin resistance in non–Hispanic blacks (7). Non–Hispanic blacks have higher levels of HDL-cholesterol (a protective cardiovascular disease factor) during adolescence and lower levels of triglycerides (a proatherogenic factor) throughout the life span (5,6). Interestingly, non–Hispanic blacks appear to have lower baseline levels of triglycerides; though these triglyceride levels increase with worsening insulin resistance, they are less likely to exceed currently used cutoff values that are heavily influenced by the normal range of triglycerides among non–Hispanic whites (7). It is possible that by the time a non–Hispanic black individual exhibits triglyceride or HDL-cholesterol abnormalities that exceed current limits for MetS classification, he or she may already have a more advanced condition of MetS that includes higher hsCRP levels than those observed in non–Hispanic whites.

To assess for this possibility, we evaluated mean values of the individual

Table 3—*Correlations* between MetS components and hsCRP†*

	BMI	Waist circumference	Systolic blood pressure	Diastolic blood pressure	Triglycerides*	HDL-cholesterol	Fasting glucose
Overall							
Non–Hispanic white	**0.53** (0.49–0.57)	**0.51** (0.46–0.55)	**0.17** (0.11–0.23)	−0.01 (−0.07 to 0.06)	**0.20** (0.14–0.26)	**−0.17** (−0.23 to −0.11)	−0.05 (−0.11 to 0.01)
Hispanic	**0.49** (0.45–0.53)	**0.50** (0.46–0.54)	**0.16** (0.11–0.21)	0.03 (−0.01 to 0.09)	**0.19** (0.14–0.24)	**−0.22** (−0.27 to −0.17)	0.05 (−0.01 to 0.10)
Non–Hispanic black	**0.55** (0.51–0.59)	**0.55** (0.51–0.59)	**0.14** (0.09–0.20)	0.02 (−0.04 to 0.08)	**0.19** (0.13–0.24)	**−0.23** (−0.28 to −0.17)	0.05 (−0.01 to 0.10)
Male							
Non–Hispanic white	**0.53** (0.46–0.59)	**0.52** (0.46–0.58)	**0.21** (0.13–0.29)	0.00 (−0.08 to 0.09)	**0.20** (0.12–0.28)	**−0.21** (−0.29 to −0.13)	0.03 (−0.05 to 0.11)
Hispanic	**0.50** (0.45–0.56)	**0.52** (0.46–0.57)	**0.18** (0.11–0.25)	0.07 (0.00–0.15)	**0.16** (0.09–0.23)	**−0.27** (−0.34 to −0.20)	0.05 (−0.02 to 0.12)
Non–Hispanic black	**0.50** (0.44–0.56)	**0.52** (0.46–0.57)	**0.16** (0.08–0.24)	0.04 (−0.04 to 0.12)	**0.17** (0.09–0.24)	**−0.24** (−0.32 to −0.17)	0.06 (−0.02 to 0.14)
Female							
Non–Hispanic white	**0.54** (0.48–0.60)	**0.52** (0.45–0.58)	**0.16** (0.07–0.24)	−0.02 (−0.11 to 0.07)	**0.21** (0.12–0.29)	**−0.15** (−0.24 to −0.06)	−0.11 (−0.20 to −0.02)
Hispanic	**0.48** (0.42–0.54)	**0.50** (0.44–0.55)	**0.17** (0.10–0.25)	−0.02 (−0.10 to 0.05)	**0.23** (0.16–0.30)	**−0.20** (−0.26 to −0.12)	0.06 (−0.01 to 0.13)
Non–Hispanic black	**0.58** (0.52–0.63)	**0.58** (0.51–0.63)	**0.22** (0.13–0.30)	−0.05 (−0.14 to 0.04)	**0.23** (0.15–0.31)	**−0.23** (−0.32 to −0.15)	0.09 (0.00–0.18)

Data are Pearson *r* (95% CI). *Correlation estimates and corresponding 95% CIs did not incorporate sampling weights provided in NHANES; significant correlations ($P < 0.05$) in bold. †Natural log of variable was used to achieve normality.

MetS components among adolescents classified as having MetS. In addition to having more hypertension, non–Hispanic black adolescents with MetS have a higher BMI than the other groups. However, no other racial/ethnic differences in MetS components were detected. Hypertension was not as significantly associated with hsCRP levels (Table 3), leaving ethnic differences in BMI as a potential explanation for the elevated hsCRP levels in non–Hispanic blacks with MetS. In many ways, this is not surprising. BMI (and waist circumference) carried by far the strongest association with hsCRP in this study (Table 3) and elsewhere (13), and these relationships have been demonstrated to be similarly strong among non–Hispanic blacks and among non–Hispanic whites and Hispanics (25).

The relationship between BMI and hsCRP may relate to fat tissue mass (25). Interestingly, for a given BMI and waist circumference, non–Hispanic blacks have less overall fat mass and more lean mass than non–Hispanic whites (24). As a result of data limitations within NHANES 1999–2008, it remains unclear whether the higher BMI among non–Hispanic black adolescents with MetS is due to higher fat mass, higher lean mass, or both. Nevertheless, racial/ethnic differences in lean mass amount to approximately 3–4% of body weight, whereas the 10% difference in BMI among non–Hispanic blacks with MetS compared with other racial/ethnic groups suggests an element of increased fat tissue as well (24). Additionally, pubertal status is known to contribute to insulin resistance and, overall, occurs earlier in non–Hispanic blacks than in other ethnicities. While pubertal stage of subjects is not reported in NHANES 1999–2008, non–Hispanic white adolescents with MetS tended to be older than the other ethnicities (Supplementary Materials). Consequently, even though MetS occurs at a lower rate in non–Hispanic black adults than in non–Hispanic whites or Hispanics, it cannot be ruled out that pubertal differences play a role.

Notably, non–Hispanic black adolescents with MetS did not have significantly different levels of triglycerides or HDL-cholesterol compared with the other racial/ethnic groups (Supplementary Materials). This suggests that whereas population differences in lipid levels are wide, non–Hispanic black adolescents with MetS have levels of triglycerides and HDL similar to those of other racial/ethnic groups. While this may represent a subset of non–Hispanic black adolescents with a greater predisposition toward dyslipidemia, these data also support the hypothesis that non–Hispanic black adolescents with MetS have a more advanced condition—potentially driven by a further degree of obesity, as represented by BMI differences. This imbalance in the classification of MetS has led the American Diabetes Association and others (6,23) to call for alternate sets of criteria for diagnosing MetS. These alternate approaches could include the use of race/ethnicity-specific cutoff values for MetS components or development of continuous criteria that add up the degree of abnormality in each of the individual components instead of requiring a person to exceed cutoff values for a set number of components. Use of such criteria could result in the earlier classification of MetS among at-risk adolescents.

In conclusion, the classification of MetS confers a greater differential in hsCRP levels among non–Hispanic black adolescents than among non–Hispanic whites but not among Hispanics. Though the exact nature of the relationships between MetS, BMI, and hsCRP between ethnicities remains unclear, these data suggest a more advanced state of inflammation in the setting of MetS among non–Hispanic black adolescents than among non–Hispanic whites, with one possible etiology being a relatively more severe condition of MetS. Given racial/ethnic differences in the classification of MetS using current criteria, there remains a great need for specific diagnostic tools to assess inflammation-related and MetS-related risk among adolescents of all racial/ethnic groups.

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M.D.D. researched data and wrote the manuscript. M.J.G. researched data and helped write the manuscript. A.E.S. researched data, contributed to discussion, and reviewed and edited the manuscript.

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